vomiting, anorexia and abdominal bloating may also occur.

The diagnosis may be made on examination of several stools. Patients not having diarrhea usually pass only cysts. Trophozoites appear only in patients having significant diarrhea. Stool examination is an insensitive means of establishing the diagnosis. The most sensitive methods are duodenal intubation, small intestine mucosal biopsy and gently smearing the mucus on the biopsy onto a slide. The slide is air-dried and stained with Giemsa stain.

The pathogenesis of symptoms in giardiasis is not defined. A number of phenomena have been observed. These include epithelial and mucosal invasion, reversible disaccharidase and vitamin B_{12} and folate malabsorption.

The organism has been shown to deconjugate bile acids in the absence of bacterial overgrowth. Patients with gastrointestinal immunodeficiency diseases have a peculiar susceptibility to Giardia infection. Diarrhea and malabsorption in these patients is almost always due to this organism.

Giardiasis should always be treated when identified, even in asymptomatic patients. Between 80 and 90 percent of infections may be cured with administration of quinacrine, 100 mg three times a day, for one week. Repeat courses are occasionally required. Metronidazole (Flagyl®), 750 mg three times a day for ten days, is an excellent alternative drug. This drug is an investigational new drug for this purpose in the United States.

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Clindamycin-Associated Colitis

REPORTS OF UNTOWARD EFFECTS from oral and intravenous therapy with clindamycin noting severe and even fatal complications have abounded in the literature in the last two years. Affected patients report abdominal cramps and watery diarrhea, with or without hematochezia or passage of mucus. These symptoms can occur within one day of therapy and as long as several months after stopping administration of the drug, as reported in a number of recent articles. Typical findings of clindamycin-associated pseudomembranous colitis at sigmoidoscopy consist of mucosal friability, with interspersed raised creamy, yellowish plaques. Findings on rectal biopsies have shown inflammatory changes with mucosal destruction, fibrinoid necrosis and a prominent pseudomembrane (pseudomembranous colitis). Colonoscopy showed these changes to extend beyond the surface routinely surveyed at sigmoidoscopy, and using barium enemas it was shown that the inflammatory changes can involve the entire colon.

What makes clindamycin-induced pseudomembranous colitis so dangerous is its frequent unresponsiveness to routine therapeutic measures. Administration of diphenoxylate hydrochloride (Lomotil®) and kaolin powders (Kaopectate®) often brings no relief and may in fact, as stated in one package insert, "prolong or worsen the condition." Use of steroids, though recommended by some, offered no significant improvement to the patients observed. Recently Burpige and Milligan have advocated the use of cholestyramine as having proved beneficial in two cases they have studied. This is welcome information and if corroborated would make cholestyramine the first useful medication against the dreaded complication, but this needs to be further documented.

The best way to reduce the incidence of clindamycin-associated colitis is to limit the use of clindamycin to its proper indications. It should never be used as a routine medication except

- As an alternative drug in patients with staphylococcus aureus infections who cannot tolerate penicillin or cephalosporin,
- In the management of suspected or proven severe infections due to Bacteroides fragilis and
- In the initial management, pending bacteriological results, in acute peritonitis following perforation of a viscus.

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